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16. ABSTRACT

Complex auditory processing deficits have been reported in children with asymptomatic lead (Pb) exposure (1,2) as well as acute Pb poisoniong (3). Hearing thresholds have not been systematically evaluated in Pb exposed children, although hearing impairments have been observed in Pb workers (4). Hearing thresholds and brainstem auditory evoked potentials (BAEPs) have been assessed in two groups of North Carolina children with blood lead (PbB) levels ranging from 6 to 59 μg/dl. Linear increases in latencies of BAEP waves III and V relative to PbB levels obtained five years earlier were found in 49 children aged 6 to 12 years old (5). Results of audiometric screening and BAEP testing in an independent group of children aged 4 to 7 years will be described below.

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EFFECTS OF LOW TO MODERATE LEAD EXPOSURE ON BRAINSTEM AUDITORY EVOKED POTENTIALS IN CHILDREN

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Introduction

Complex auditory processing deficits have been reported in children with asymptomatic lead (Pb) exposure (1,2) as well as acute Pb poisoning (3). Hearing thresholds have not been systematically evaluated in Pb exposed children, although hearing impairments have been observed in Pb workers (4). Hearing thresholds and brainstem auditory evoked potentials (BAEPs) have been assessed in two groups of North Carolina children with blood lead (PbB) levels ranging from 6 to 59 µg/dl. Linear increases in latencies of BAEP waves III and V relative to PbB levels obtained five years earlier were found in 49 children aged 6 to 12 years old (5). Results of audiometric screening and BAEP testing in an independent group of children aged 4 to 7 years will be described below.

Methods

Eighty Black children aged three to seven, from high risk neighborhoods for exposure to lead based paint chips, were referred for study by county health agencies. The seventy-five children who passed the screening process came from low income families (class IV or V on Hollingshead Scale), were healthy with no history of perinatal complication or neurological disorder, and were asymptomatic with regard to Pb poisoning. A brief medical and neurological examination was made by a pediatrician prior to BAEP testing. Ears were checked by otoscope or tympanograph for otitis media. Children with ear infections or other acute illnesses were treated and rescheduled.

Venipuncture blood samples were obtained on the day of testing. PbB level was determined by the modified Delves cup atomic absorpting procedure (6). Prior PbB measures were obtained from county health records of children when available.

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Hearing threshold a 2 KHz pure tone was determined audiometrically before BAEP testing. If the reliability of the behavioral threshold was questionable, electrophysiological hearing threshold was determined using clicks presented at 33 Hz. A descending series of monaural BAEPs was obtained with threshold defined as the intensity at which BAEP wave V could no longer be seen. The BAEP was recorded at a bandpass of 100-3000 Hz and sampled at 50 KHz. Two replicate averages of 2000 binaural clicks of alternating polarity were obtained using a Cz to linked mastoid derivation. Clicks were presented at 60 dB above hearing threshold using a stimulus rate of 11.1 clicks/sec. Latency measures of upward deflections of waves I, III, and V were made. A vertex-positive or mastoid-negative event gave an upward deflection. Central conduction times (I-III, III-V, and I-V interpeak latencies [IPL]) were also calculated.

A multiple regression model was used with PbB as predictor variable, BAEP latencies and IPLs as outcome variables, and age, gender, socioeconomic status (SES), quality of the home caregiving environment, and maternal IQ as control variables. SES was determined by the Hollingshead Two Factor Index. Caregiver practices were measured with a standardized HOME checklist (7). Maternal IQ was obtained with the Peabody Picture Vocabulary Test (1983 revised). Child IQ was also measured with the Stanford Binet Intelligence Test (1972 revised) for use as an outcome variable. A stepwise approach was used with potentially confounding interactions (age x BAEP, gender x BAEP, etc.) tested first, then main effects of control variables, and finally the main effect of PbB. The confirmatory test was limited to wave III and V latencies, based on previous findings (5), using current PbB levels. Exploratory analyses were also conducted using maximal PbB values obtained from medical histories in a polynomial model including linear, quadratic and cubic components of outcome variables. Demographic characteristics of the sample are summarized in Table 1.

Table 1. Demographic characteristics of study sample.

VARIABLE	N	MEAN	SD	MIN	MAX
AGE (months)	75	58.4	11.7	33	94
SES	75	70.8	6.2	55	84
CHILD IQ	75	86.1	11.7	60	119
MATERNAL IQ	75	63.4	11.3	40	94
HOME	75	59.8	15.1	20	87
CURRENT PbB (ug/dl)	75	20.6	9.7	6.2	47.4
MAXIMUM PbB	75	26.7	14.1	6.2	56.0

Results

Hearing thresholds as shown in Fig. 1 increased linearly with maximal PbB [$F(1,55) = 13.0$, $p = .0007$, $R = .19$]. The confirmatory analysis of wave III and V latencies failed to show any linear relationship to current PbB. Exploratory polynomial analyses of linear, quadratic and cubic components of the relation ships between PbB and BAEP measures were therefore undertaken. Results of these analyses using maximal PbB values are shown in Fig. 2 and Table 2. Wave I latency decreased linearly, while IPL I-V increased directly with maximal PbB. Significant curvilinear relationships of PbB, wave III and V latencies and IPLs I-III and IV were also found. That is, these latencies and IPLs tended to decrease below 25 µg/dl, but increase above 25 µg/dl.

Discussion

Elevated hearing thresholds have been reported in Pb workers (4), but changes in hearing thresholds have not been reported previously in Pb-exposed children. This exploratory finding is of considerable interest in view of the well-documented association of childhood lead poisoning, mental retardation, and more subtle learning disabilities. A selective hearing loss occurring in early childhood that remains undetected could result in speech pathology and a variety of learning disorders as the child grows. Such problems frequently are not identified until the child reaches school age and demonstrates performance deficiencies.

On the other hand, the preliminary nature of the observed relationship between PbB level and hearing threshold must be emphasized. No such relationship was found in older children (5) in whom the same method was used to estimate hearing threshold. In order to evaluate the validity and generality of the present observation, a comprehensive audiometric assessment of an independent sample of children at risk for lead poisoning should be undertaken.

The linear decrease of wave I latency with increasing PbB is inconsistent with the apparent increase in hearing threshold. One possibility is that no hearing deficit existed in these children and that wave I latency simply reflected changes in absolute click intensity. If this were the case, however, parallel changes in wave III and V latencies would be expected, but were not found. Another possible explanation is that Pb exposure may alter a higher level of auditory perception associated with

Table 2. Linear, quadratic and cubic trends in the relationship of maximal PbB and BAEP measures: p-values

Variable	Polynomial	Linear	Quadratic	Cubic
Latency I (MSEC)	.013	.007	.14	.21
Latency III	.021	.41	.003	.50
Latency V	.015	.75	.001	.79
IPL I-III	.06	.15	.022	.76
IPL I-V	.010	.038	.008	.62
IPL III-V	.09	.08	.08	.64

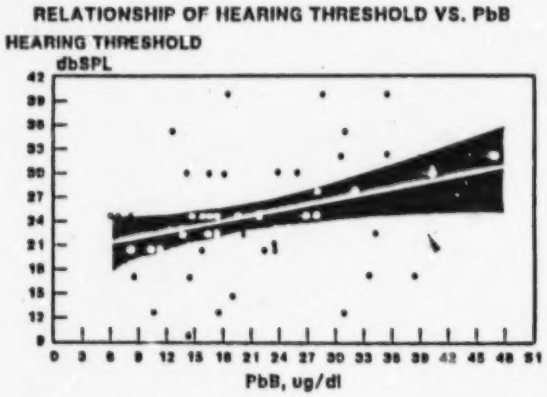

Figure 1

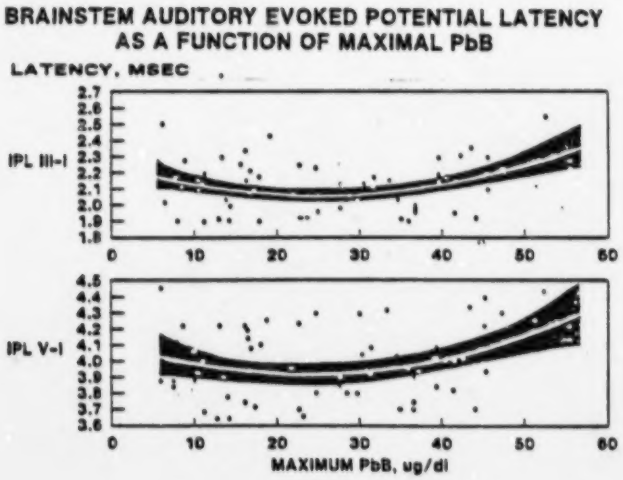

Figure 2

psychophysical judgment of loudness rather than simple hearing threshold. This hypothesis could be tested by systematic comparison of audiometric thresholds (dependent on judgment) and electrophysiological thresholds (independent of judgment).

The curvilinear relationships of wave III and V latencies, interpeak latencies I-III and I-V and PbB are puzzling. A simple linear increase of wave III and V latencies relative to Pb exposure history was observed in the five-year follow-up study of older children. Decreasing latencies in the 6-24 µg/dl range followed by increasing latencies above 25 suggest the possibility of a biphasic effect of Pb on nerve conduction in the auditory pathway. Ewert et al. (8) reported a similar paradoxical decrease in visual evoked potential (P1) latency and peripheral nerve conduction velocity in children with PbB levels of 4-23 µg/dl.

Although the adjustment of stimulus intensity to achieve equal sensation level across subjects is a widely used procedure in psychophysical research, this approach is problematic in BAEP testing. Wave I latency is extremely sensitive to stimulus intensity and hearing threshold, but the latencies of later waves are differentially influenced by these factors. Interpeak latencies, however, are relatively immune to differences in hearing thresholds (peripheral auditory effects). The linear increase in I-V interpeak latency with increasing maximal PbB is the most straightforward evidence of an adverse effect of Pb exposure on auditory function. This evidence supports our earlier suggestion (5) that Pb affects central conduction time in the auditory pathway. Since these findings derive from post hoc exploratory analyses, however, they must be interpreted cautiously pending replication and clarification.

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